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The contribution of previous lameness events and body condition score to the occurrence of lameness in dairy herds: A study of 2 herds

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ABSTRACT

It has been demonstrated that low body condition and previous occurrence of lameness increase the risk of future lameness in dairy cows. To date the population attributable fraction (PAF), which provides an estimate of the contribution that a risk factor makes toward the total number of disease events in a population, has not been explored for lameness using longitudinal data with repeated measures. Estimation of PAF helps to identify control measures that could lead to the largest improvements on-farm. The aim of this study was to use longitudinal data to evaluate the proportion of lameness that could be avoided in 2 separate herds (2 populations) through (1) reduced recurrence of previous lameness events, (2) and moving body condition score (BCS) into more optimal ranges. Data were obtained from 2 UK dairy herds: herd A, a 200-cow herd with 8 yr of data from a total of 724 cows where lameness events were based on weekly locomotion scores (LS; 1 to 5 scale), and herd B, a 600-cow herd with data recorded over 44 mo from a total of 1,040 cows where treatment of clinical cases was used to identify lameness events. The PAF for categories of BCS were estimated using a closed equation appropriate for multiple exposure categories. Simulation models were used to explore theoretical scenarios to reflect changes in BCS and recurrence of previous lameness events in each herd. For herd A, 21.5% of the total risk periods (cow-weeks) contained a lameness event (LS 3, 4, or 5), 96% of which were repeat events and 19% were recorded with BCS <2 (3 wk previously; 0 to 5 scale). When lameness events were based on 2 consecutive weeks of LS 4 or 5, 4% of risk periods were recorded as lame, of which 89.5% were repeat events. For herd B, 16.3% of the total risk periods (consecutive 30 d) contained a lameness event (72.6% were repeat events) and 20% were recorded with BCS ≤2 (0 to 120 d previously).

The median PAF for all previous lameness was between 79 and 83% in the 2 herds. Between 9 and 21% of lameness events could be attributed to previous lameness occurring >16 wk before a risk period. The median PAF estimated for changes in BCS were in the region of 4 to 11%, depending on severity of lameness. Repeated bouts of lameness made a very large contribution to the total number of lameness events. This could either be because certain cows are initially susceptible and remain susceptible, due to the increased risk associated with previous lameness events, or due to interactions with environmental factors. This area requires further research.

Key words: lameness, dairy cattle, population attributable fraction, body condition score, previous lameness events

INTRODUCTION

Numerous risk factors for lameness in dairy cattle have been reported in the literature, including risk factors related to the external environment such as flooring surfaces and time spent standing (Galindo and Broom, 2000; Bergsten et al., 2015) as well as animal-based factors that might affect structure and function of the claw such as milk yield, BCS, and previous lameness events (Green et al., 2014; Randall et al., 2015). Low BCS and previous lameness are both risk factors for lameness that occur repeatedly over time and have been highlighted as important for lameness control (Hirst et al., 2002; Bicalho et al., 2009; Green et al., 2014; Randall et al., 2015, 2016). Randall et al. (2015) showed that relatively low body condition precedes and is associated with an increased risk of a first lameness event in a cow's life. Consequently, management strategies to maintain appropriate BCS may provide an opportunity for the dairy industry to reduce lameness in herds. Hirst et al. (2002) demonstrated that dairy heifers with lameness-causing claw horn lesions were at greater risk of lameness in subsequent lactations. A recent study suggested that this relationship might be

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explained by development of new bone (exostosis) on the distal phalanx (Newsome et al., 2016). If this is an irreparable anatomical change to the foot, it would contribute toward an increased risk of a cow becoming lame again. Odds ratios (**OR**) reported for these 2 risk factors indicate that they are highly associated with lameness; for example, the OR associated with moving from a nonlame to a lame state for cows with BCS 1.00 to 1.75 at calving versus 2.50 to 2.75 was 7.73 (2.37–17.71) and the OR associated with clinical lameness for cows having been identified lame 31 to 60 d previously versus no previous lameness was 13.80 (10.58–17.78; Green et al., 2014; Lim et al., 2015).

The population attributable fraction (**PAF**) provides an estimate of the contribution that a risk factor makes to the total disease burden in a population. Knowledge of the PAF of risk factors can facilitate decision making for farmers and policy makers to maximize disease reduction with existing resources when the knowhow exists, or it can influence funders of research (Steenland and Armstrong, 2006) when knowledge to reduce the effect of risk factors is not known.

A range of formulas is used to calculate PAF and these have different limitations, such as biases arising when adjusted estimates of relative risk (**RR**) are used or when the exposure is across different levels (Rockhill et al., 1998; Benichou, 2001; Steenland and Armstrong, 2006). Where risk factors vary over time, the method used to estimate PAF must account for repeated risk events. In addition, a risk factor can be complex; for example, cows in a herd have a range of body conditions rather than a uniform BCS of, for example, 3, so assessing a change in BCS to reduce the PAF needs to use a continuous scale for BCS. Simulation can be used to estimate PAF to allow for sources of uncertainty, such as uncontrolled confounding, to be incorporated into estimates (Steenland and Armstrong, 2006) as well as allowing for more complex scenarios to be investigated (Hudson et al., 2014).

The aim of this study was to investigate the contribution of previous lameness and BCS to the occurrence of total lameness events in 2 UK dairy herds. A novel simulation-based approach to estimating PAF for lameness risk factors was used.

MATERIALS AND METHODS

Study Herds

Data were obtained from 2 UK dairy herds, where detailed and accurate herd records were available. Study herds and data sets have been described in detail by Green et al. (2014) and Randall et al. (2015). They are summarized here briefly.

Herd A. A total of 724 Holstein Friesian dairy cows managed on the Langhill herd held at the Scotland's Rural College's Crichton Royal research farm, Dumfries, Scotland, with data recorded over an 8-yr period from 2003 to 2011 (Randall et al., 2015). Cows were managed on a long-term 2×2 factorial genetic and feeding system study; select and control genetic lines (Pryce et al., 1999) were divided equally into low-forage (**LF**) and high-forage (**HF**) groups and managed as 1 herd of approximately 200 cows, as described in detail by Chagunda et al. (2009). The LF cows were continuously housed whereas HF cows were grazed during the summer grazing period (typically March to November). Cows were milked 3 times daily and the herd was all-year-round calving. Target yields were 13,000 and 7,500 kg per cow per year for LF and HF cows, respectively. Housing was the same for LF and HF cows: cubicles with mattresses and automatically scraped grooved concrete passageways. Regular footbathing was carried out and a professional foot trimmer attended the whole herd twice a year. Locomotion scores (**LS**) were recorded weekly by trained assessors on a 1 to 5 scale (Manson and Leaver, 1988). Lame cows (LS 4 or 5 on a single occasion or 2 successive scores of LS 3) were treated by a veterinarian on a weekly basis before 2006 and every 2 wk after this time. Severely lame cows were treated within 24 h by trained farm staff. The BCS was measured weekly using a 0 to 5 scale with increments of 0.25 (Mulvany, 1977). All health, production, and management data were recorded in a database.

Herd B. A total of 1,040 Holstein dairy cows on 1 dairy farm in Somerset, England, with data recorded over 44 mo between 2008 and 2011 (Green et al., 2014). Cows were milked twice daily in a 60-point rotary parlor and continuously housed all year, apart from summer when grazed during the last 2 mo of lactation. Rations were formulated with the aim of maximizing yield while minimizing feed costs and fed to milking cow groups (early, mid, and late lactation) accordingly. Biotin was added at 20 mg/cow per day. Housing was modern free-stall accommodation with water mattresses in cubicles and solid concrete passageways with automatic scrapers. Mean yearly yield was approximately 10,000 kg per cow per annum. A professional foot trimmer attended the herd each month; typically cows at the end of lactation and with misshapen feet were trimmed, with a minimum routine foot trim once per year. Daily observations of the herd by senior herdsmen identified lame cows, which were treated under veterinary direction using standard protocols, generally within 2 to 3 d. Body condition score was recorded at 60-d intervals throughout the study period by the head herdsman with appropriate training to prevent drift in scoring, on a scale of 0 to 5 in 0.5 increments [based on examina-

tion of the transverse processes of the lumbar vertebrae, the ribs, ischial tuberosity, ligaments of the pelvis, and surrounding fat (Green et al., 2014)]. Health, production, BCS, and lameness treatments were recorded in Interherd (National Milk Records).

Statistical Analysis

To account for the longitudinal nature of the data, risk factors where events varied at repeated measurements were lagged (e.g., BCS, previous lameness, and milk yield) and frailty models were constructed to take into account repeated measures of the outcome (lameness events). The main difference between herd A and herd B was in defining lameness events; herd A was based on weekly locomotion scoring, whereas herd B was based on treatment for lameness from the farmer's records. For herd A, 2 separate definitions for a lameness event were investigated; these were (1) 1 wk with LS 3, 4, or 5 (less severe lameness), and (2) 2 consecutive weeks with LS 4 or 5 (more severe lameness). There were 3 stages to estimating the PAF: (1) constructing models to estimate adjusted RR for BCS and previous lameness, (2) estimating PAF for BCS categories to compare estimates using closed equation and simulation approaches, and (3) using simulation to estimate PAF for changes in BCS and occurrence of previous lameness within the 2 herds to quantify the contribution of these risk factors to total lameness in each herd.

The annual incidence rate of lameness was calculated as (number of new lameness events divided by number of cow-weeks at risk) multiplied by 52 for herd A and (number of new lameness events divided by number of cow-months at risk) multiplied by 12 for herd B. For herd A, the weekly incidence rates over the study period were calculated as the number of new lameness events divided by the number of cows eligible (i.e., those cows not lame in the previous risk period) and prevalence was calculated as number of lameness events divided by number of observations.

Stage 1: General Approach to Modeling; Estimating Coefficients for Previous Lameness and BCS

Data handling and model construction are described in detail by Randall et al. (2015) for herd A and Green et al. (2014) for herd B.

Binary outcomes investigated for herd A were LS 3, 4, or 5 in 1 wk (model 1a) and LS 4 or 5 over 2 consecutive weeks (model 1b). The model outcome in herd B was also binary: yes/no for treatment of lameness (all causes included sole hemorrhage, sole ulcer/white line disease, and digital dermatitis; model 2). Mixed effects

logistic regression models were constructed in MLWin 2.28 (Rabash et al., 2009). Where possible, missing observations were included as a categorical variable and fitted within the models to minimize loss of data. Initial parameter estimation for model parameters was carried out by iterative generalized least square procedures (Goldstein, 2003) and using forward selection of explanatory variables; explanatory variables were left in the model if the 95% credible interval of the OR did not include unity. Final parameter estimates were made using Markov chain Monte Carlo (MCMC) to reduce biased estimates (Rabash et al. 2009), using procedures previously described by Green et al. (2004). A burn-in of 1,000 iterations was used, with final parameter estimates being based on a minimum further 9,000 iterations. Chain mixing and stability were assessed visually.

Models took the form

$$\text{Lame}_{ij} \sim \text{Bernoulli}(\text{probability} = \pi_{ij})$$

$$\text{Logit}(\pi_{ij}) = \alpha + \beta_1 \mathbf{X}_{ij} + \beta_2 \mathbf{X}_j + u_j$$

$$[u_j] \sim N(0, \sigma_v^2),$$

where subscripts i and j denote the i th observation of the j th cow, respectively; π_{ij} = probability of a lame outcome for the i th observation of the j th cow; α = intercept value; β_1 = vector of coefficients for \mathbf{X}_{ij} (herd A included logarithm of the week of the study up to the power 3); \mathbf{X}_{ij} = vector of covariates associated with each observation; β_2 = coefficients for covariates \mathbf{X}_j ; \mathbf{X}_j = vector of covariates associated with each cow; and u_j = random effect to account for residual variation between cows (assumed to be normally distributed with mean = 0 and variance = σ_v^2) and residual error.

Explanatory variables included in the models for herd A were weeks in milk, week of the study, parity (categorical 1 to 4+), age at first calving (categorical <24, 24 to 27, 28 to 30, 31 to 33, and greater than 33 mo), BCS change 0 to 4 wk postcalving (categorized as 0 = loss, 1 = no change, 2 = gain), BW (categorical <550, 550 to 700, and >700 kg), assessor of locomotion and body condition, feed – genetic group, and milk yield 16 wk previously (average daily kg per week; categorical <12, 12 to 24, 25 to 37, 38 to 50, and >50 kg). Variables of interest were time since previous lameness (categorized in 4-wk intervals from time t to >16 wk) and BCS (categorical <2, 2, 2.25, 2.5, 2.75, 3, and >3). Explanatory variables included in the model for herd B were parity (categorical 1–6+), year quarter, month in herd, DIM (at the end of a 30-d period), milk yield (kg per day) measured at the most recent monthly milk recording, and yield lagged by 1 mo. Variables of

interest were time since previous lameness event (data were available from 2002, categorized in 30-d intervals from time t to > 120 d) and BCS >2 lagged by 0 to 2 mo and 2 to 4 mo.

Posterior predictions were used to assess model fit by visual comparison to the observed data (Gelman et al., 1996). Standardized residuals at the cow level (level 2) were also assessed for normality (Rabash et al., 2009). The Hosmer-Lemeshow test (Hosmer and Lemeshow, 1989) was used as a statistical test for goodness of fit. Cow-level residuals were found to be overdispersed and nonnormal for models 1a and 1b; therefore, random effects were removed, which improved model fit such that it was very good without random effects, and were used as the final models.

Descriptive Statistics and Results from Modeling

Herd A. Of the 724 cows ever in herd A, 674 (93.0%) had at least 1 wk with LS 3, 4, or 5 and 375 (51.8%) had at least 1 lameness event with LS 4 or 5 for 2 consecutive weeks. There were a total of 79,565 and 78,698 cow weeks at risk in models 1a and 1b, respectively. The number of lameness events were 17,114 and 3,572, respectively, for models 1a and 1b. The annual incidence rate of lameness was 7.4 cases per cow-year when a lameness event was one week LS 3, 4, or 5 and 0.7 cases per cow-year when a lameness event was 2 consecutive weeks LS 4 or 5. The weekly incidence rates over the study period are shown in Figure 1 for models 1a (one-week LS 3, 4, or 5) and 1b (2 consecutive weeks LS 4 or 5), respectively. Figure 1 also shows the prevalence for each week of the study period for models 1a (one-week with LS 3, 4, or 5) and 1b (2 consecutive weeks LS 4 or 5), respectively. Both weekly incidence rates and prevalence of LS 3, 4, or 5 increased during the second half of the study period for herd A. Figure 2 shows the frequency distribution for number of consecutive weeks with LS 3, 4, or 5 and LS 4 or 5, respectively, demonstrating that the majority of lameness events had a duration of 1 wk. The median BCS was 2.25 (range, 0.75 to 4.25) for herd A. The proportion of the cow-week risk periods exposed to BCS categories <2 , 2, 2.25 and 3 were 0.19, 0.23 (0.24 for model 1b), 0.26 and 0.05, respectively, for models 1a and 1b. The proportion of observations where there was a previous lameness event in the 1 to 4 wk prior was 0.4; 5 to 8 wk was 0.38; 9 to 12 wk was 0.36; 13 to 16 wk was 0.34; and >16 wk was 0.73 for model 1a. For model 1b the proportion of observations where there was a previous lameness event in the 1 to 4 wk prior was 0.079; 5 to 8 wk was 0.074; 9 to 12 wk was 0.070; 13 to 16 wk was 0.067; and >16 wk was 0.29. Odds ratios and 95% credible intervals from models 1a and 1b for BCS

and previous lameness are reported in Table 1. For all other covariates included in the final model, parameter values and significance were similar to those previously reported (Randall et al., 2015). Assessment of model fit was considered good. For model 1a, BCS categories 3 wk previously were significantly associated with the lameness outcome $LS \geq 3$. Body condition score = 3, 3 wk previously had the lowest OR (i.e., the lowest risk of lameness) and therefore was used as the baseline category for simulations described below in stage 2. A BCS <2 had the highest OR [95% credible interval = 1.29 (1.15 to 1.45)] compared with the baseline category. Previous lameness variables were also significant; lameness in the previous 1 to 4 wk compared with no previous lameness had the highest OR (95% credible interval = 3.65 (3.48 to 3.83)). For model 1b, BCS 1 wk previously had the largest effect size and therefore was left in the final model. As for model 1a, BCS = 3 had the lowest OR and was used as the baseline category for simulations in stage 2. Body condition score <2 had the highest OR compared with the baseline category BCS = 3 [OR (95% credible interval) = 1.66 (1.27 to 2.16)]. Previous lameness variables were also associated with a significantly increased risk of lameness; lameness in the previous 1 to 4 wk had the highest OR [95% credible interval = 18.72 (16.97 to 20.66)] compared with no previous lameness.

Herd B. A total of 14,530 risk periods were obtained from 1,040 cows from herd B and the mean number of observations was 10 (range 1–36) per cow. The annual incidence rate for the study period was 1.4 cases per cow-year. A total of 14,461 BCS were included in the data set; the median BCS was 2.5 (range: 1 to 5). In total, 647 cows were treated for lameness; the proportion of observations where there was exposure to previous lameness 1 to 30 d ago, 31 to 60 d ago, 61 to 90 d ago, 91 to 120 d ago, and greater than 120 d ago were 0.21, 0.10, 0.05, 0.04, and 0.17, respectively. Of the 1,040 cows, 62.2% were ever lame during the study. Odds ratios and 95% credible intervals from model 2 for the explanatory variables of interest (BCS and previous lameness) are reported in Table 1. For all other covariates included in the final model, parameter values and significance have previously been reported (Green et al., 2014). For model 2, BCS >2 in the last 0 to 2 mo or 2 to 4 mo was associated with a decreased risk of lameness (all causes; sole hemorrhage, sole ulcer/white line disease, and digital dermatitis) compared with BCS ≤ 2 ; OR (95% credible interval) = 0.63 (0.55 to 0.73) and 0.74 (0.60 to 0.90), respectively. All previous lameness categories were associated with an increased risk of lameness compared with no previous lameness; previously lame 1 to 30 d ago had the highest OR [credible interval = 19.69 (15.70 to 24.69)].

Stage 2: Comparing Closed and Simulation-Based Approaches to Estimating PAF

Exposure to BCS categories for each of the herds' data was used to estimate PAF using a closed formula and simulation.

A formula for multiple exposure categories described by Hanley (2001) was used for the closed method:

$$\text{PAF} = \frac{P_1 \{RR_1 - 1\} + P_2 \{RR_2 - 1\}}{1 + P_1 \{RR_1 - 1\} + P_2 \{RR_2 - 1\}},$$

where P = prevalence of exposure, and RR is calculated from the coefficients estimated for each BCS category from models 1a, 1b, and 2.

The simulation approach used posterior predictions of the number of lameness events to estimate PAF (Gelman, 2000). Models 1a, 1b, and 2 were imported into OpenBUGS version 3.2.3 (Lunn et al., 2009) alongside raw data from the respective herds. Coefficients were estimated from the models using MCMC and a burn-in of 4,000 iterations and a further 6,000 iterations for

final parameter estimates based on visual inspection of chain mixing and stability. The number of lameness events were predicted from models 1a, 1b, and 2 for the herd raw data (baseline exposure) and with exposure to each of the BCS categories sequentially removed (i.e., coefficients equal to 0). The posterior prediction for PAF was calculated as the difference in number of lameness events with and without exposure to each BCS category as a proportion of the total number of lameness events occurring in the herd. The PAF are reported only for the BCS categories that had a significant association with the outcome (lameness events).

Stage 3: Estimating PAF for BCS and Previous Lameness

Simulation was used to explore more complex scenarios by quantifying the contribution that BCS and previous lameness made toward the total number of lameness events within each herd. Scenarios explored are summarized in Table 2. Models 1a, 1b, and 2 were imported into OpenBUGS alongside raw data from

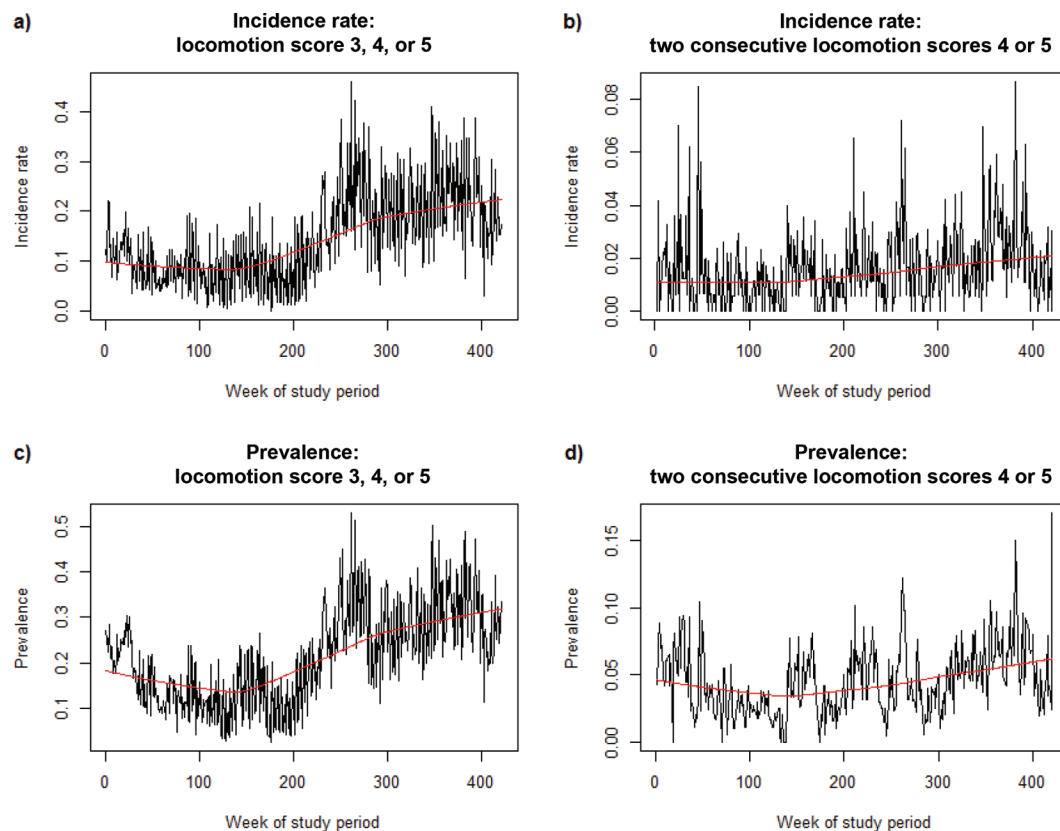


Figure 1. Weekly lameness incidence rate and prevalence over 421 wk of the study period 2003 to 2011 for herd A, 724 cows held at the Scotland's Rural College Research and Innovation Centre. The black line shows the weekly incidence rate and the middle (red) line shows locally weighted linear regression line created using the lowess function in R (R Core Team, 2016). In (a) and (c), a lameness event is defined as locomotion score 3, 4, or 5. In (b) and (d), a lameness event is defined as 2 consecutive weeks of score 4 or 5. Color version available online.

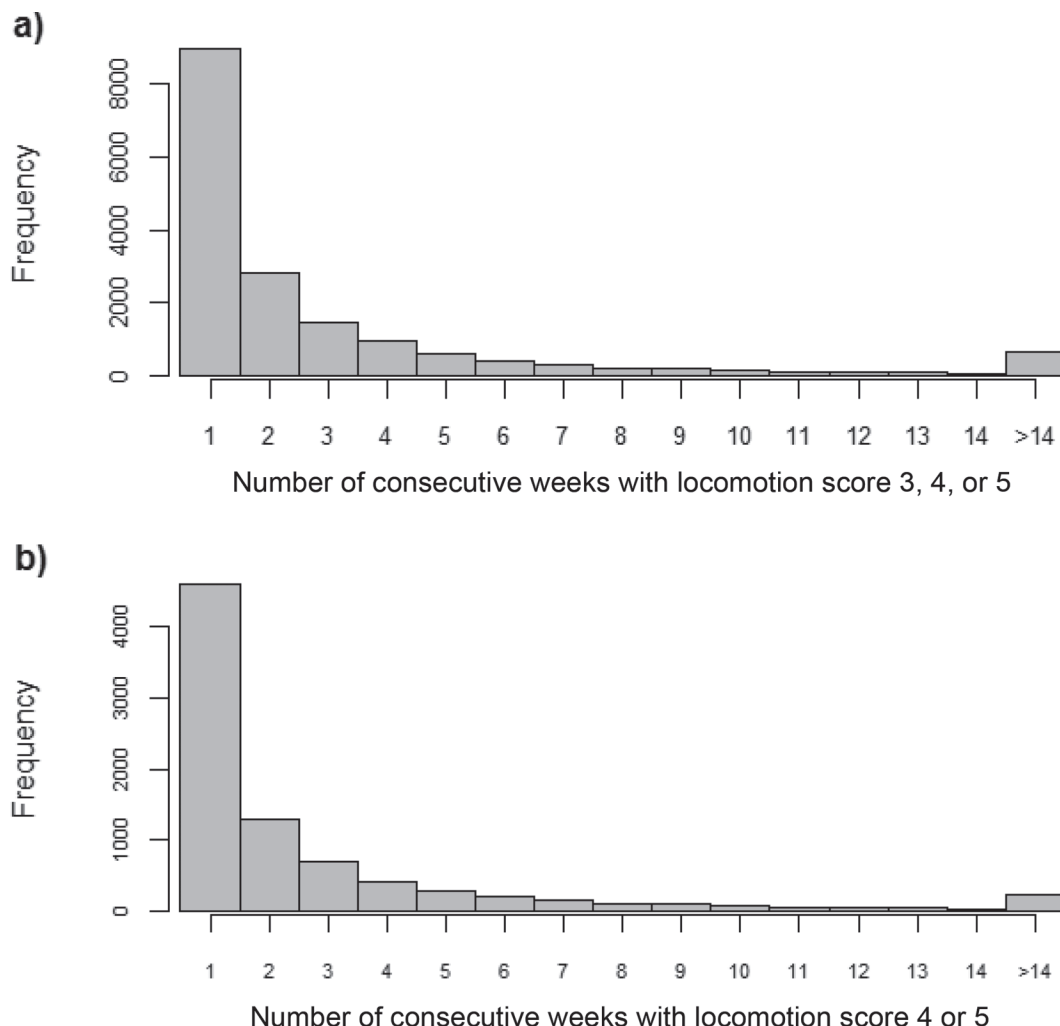


Figure 2. Frequency distributions showing the number of consecutive weeks that cows were locomotion scored as 3, 4, or 5 (a) and 4 or 5 (b) in herd A, 724 cows held at the Scotland's Rural College Research and Innovation Centre over the study period 2003 to 2011.

respective herds. For the BCS scenarios, additional categories were created for 0.5 added to the BCS score for each cow with BCS <3 for each week in the herd (i.e., 0.5 BCS gain across the whole herd apart from cows with BCS 3 or above) and 0.5 taken away from each BCS score (i.e., 0.5 BCS loss across the whole herd) in the herd A data set; data were imported to OpenBUGS. Exposure distributions for the BCS categories are summarized in Table 3. Coefficients were estimated from the models using MCMC and a burn-in of 4,000 iterations and a further 6,000 iterations for final parameter estimates, based on visual inspection of chain mixing and stability. The number of lameness events for the herd exposed to each of the scenarios (0.5 BCS gain and 0.5 BCS loss) and not exposed to these distributions (i.e., the BCS distribution of the raw herd data as a baseline) were predicted. Posterior predictions for PAF were calculated as the difference in

number of lameness events as a proportion of the total number of lameness events. For the previous lameness scenario, the raw herd data were used as the baseline scenario, with exposure to previous lameness removed for the altered scenario (i.e., all coefficients for previous lameness categories equal to zero). The number of lameness events for the herd exposed and unexposed to previous lameness events were predicted. Posterior predictions for PAF were calculated as the difference in the number of lameness events as a proportion of the total number of lameness events.

To remove the effect of lameness that occurred just before a risk period and explore only the effect of lameness events that occurred earlier, models 1a, 1b, and 2 were used to estimate PAF of lameness events that occurred a minimum of 5 wk before a current case and a minimum of 16 wk before a current case. For models 1a and 1b, the effect of previous lameness events that

occurred in the 4 to 8, 9 to 12, 13 to 16, and >16 wk previously were investigated. As these were separate covariates in the model, to investigate their effect, the

relevant coefficients were set to equal zero, where the baseline was no previous lameness in that time period. For model 2, the effects of previous lameness events

Table 1. Results of model 1a, 1b, and 2 for explanatory variables BCS and previous lameness using data obtained from the Scotland's Rural College Research and Innovation Centre dairy herd (model 1a and 1b) and a 600-cow herd in Somerset, UK (model 2)¹

Variable	N ²	Odds ratio	Lower 95% CrI ³	Upper 95% CrI
Model 1a; outcome = 1 wk with LS 3, 4, or 5 (total n = 79,565)				
BCS 3 wk previously				
3	3,612	Baseline		
<2	14,762	1.29	1.15	1.45
2	18,603	1.14	1.02	1.27
2.25	20,711	1.11	1.00	1.23
2.5	11,444	1.07	0.96	1.19
2.75	4,385	1.03	0.91	1.16
>3	2,046	1.05	0.91	1.22
Previous lameness (LS 3, 4, or 5)				
None	38,133	Baseline		
1 to 4 wk	31,483	3.65	3.48	3.83
None	3,672	Baseline		
5 to 8 wk	30,041	2.15	2.05	2.27
None	35,636	Baseline		
9 to 12 wk	28,687	1.64	1.53	1.77
None	34,547	Baseline		
13 to 16 wk	27,373	1.52	1.44	1.59
None	12,218	Baseline		
>16 wk	57,690	1.21	1.12	1.31
Model 1b; outcome = 2 consecutive weeks with LS 4 or 5 (total n = 78,698)				
BCS 1 wk previously				
3	3,718	Baseline		
<2	15,122	1.66	1.27	2.16
2	18,910	1.44	1.11	1.87
2.25	20,990	1.29	1.00	1.66
2.5	11,632	1.06	0.82	1.37
2.75	4,481	1.11	0.83	1.48
>3	2,119	1.16	0.84	1.61
Previous lameness (2 consecutive LS 4 or 5)				
None	67,770	Baseline		
1 to 4 wk	6,181	18.72	16.97	20.66
None	65,262	Baseline		
5 to 8 wk	5,812	1.99	1.78	2.22
None	62,901	Baseline		
9 to 12 wk	5,517	1.51	1.34	1.69
None	60,682	Baseline		
13 to 16 wk	5,245	1.48	1.32	1.67
None	46,587	Baseline		
>16 wk	23,064	1.62	1.46	1.79
Model 2; outcome = all causes of lameness (SH, SU/WLD, and DD) ⁴				
BCS				
BCS >2 last 0 to 2 m		0.63	0.55	0.73
BCS >2 last 2 to 4 m		0.74	0.60	0.90
Previous lameness				
None		Baseline		
1 to 30 d ago		19.69	15.70	24.69
31 to 60 d ago		13.75	10.72	17.64
61 to 90 d ago		14.51	10.76	19.58
91 to 120 d ago		13.99	10.08	19.40
>120 d ago		16.02	12.50	20.53

¹Only coefficients for explanatory variables BCS and previous lameness are reported here. Other covariates tested were found to be significant as reported by Green et al. (2014) and Randall et al. (2015). LS = locomotion score.

²N = number of observations.

³CrI = credible interval.

⁴SH = sole hemorrhage; SU/WLD = sole ulcer/white line disease; DD = digital dermatitis.

Table 2. Description of scenarios investigated for 2 UK dairy herds described by Green et al. (2014) and Randall et al. (2015)

Herd	Model	Outcome (interval)	Scenario	Description
A	1a	LS ¹ 3, 4, or 5 (weekly)	BCS gain	Whole herd gains 0.5 BCS if <3
			BCS loss	Whole herd loses 0.5 BCS
	1b	2 consecutive LS 4 or 5 (weekly)	No previous lameness	Effect of all previous lameness events removed
			BCS gain	Whole herd gains 0.5 BCS if <3
B	2	Clinical lameness; all causes (30 d)	BCS loss	Whole herd loses 0.5 BCS
			No previous lameness	Effect of all previous lameness events removed
			No previous lameness	Effect of all previous lameness events removed

¹LS = locomotion score.

that occurred in the previous 31 to 60, 61 to 90, 90 to 120, and >120 d were investigated. These were included as categories for the explanatory variable previous lameness where the baseline was none. Coefficients for the weeks being investigated were set to equal zero. The analyses were repeated as described above.

RESULTS

Comparison of Closed and Simulation Approach for Estimating PAF

The PAF estimated using both closed and simulation methods are presented in Table 4.

Model 1a. Using closed calculation methods, PAF for exposure to each BCS category were 4.49% for BCS <2 3 wk previously, 2.66% for BCS 2, and 2.38% for BCS 2.25 (total = 9.53%). The median (95% credible

interval) PAF predicted using simulation were 3.10% (1.71–4.54), 1.73% (0.14–3.33), and 1.50% (–1.31–3.13) for BCS <2, 2, and 2.25, respectively.

Model 1b. The PAF for exposure to BCS categories calculated using the closed method were as follows: 8.90% for BCS <2 one week previously, 6.00% for BCS = 2 and 4.68% for BCS = 2.25 (total = 19.58%). Using simulation, the median (95% credible interval) predicted PAF for BCS categories <2, 2, and 2.25 were 7.64% (2.81–11.23%), 5.58% (1.05–9.18%), and 3.93% (–0.76–7.74%), respectively.

Model 2. The PAF for BCS categories calculated using closed method were 9.83% for BCS <2 in the 1 to 60 d previously and 5.92% for BCS <2 in the 61 to 120 d previously. Median PAF (95% credible interval) predicted using simulation was 7.49% (4.03–10.78) and 4.28% (0.64–7.72%) for BCS <2 in the 1 to 60 d previously and 61 to 120 d previously, respectively.

Table 3. Proportion of observation in each BCS category for scenarios investigated for herd A; 724 cows held at the Scotland's Rural College Research and Innovation Centre¹

BCS category	Baseline		BCS gain		BCS loss	
	No. of observations	Proportion	No. of observations	Proportion	No. of observations	Proportion
Model 1a; total observations = 79,565						
<2	14,762	0.19	1,323	0.02	54,076	0.68
2	18,603	0.23	4,121	0.05	11,444	0.14
2.25	20,711	0.26	9,318	0.12	4,385	0.06
2.5	11,444	0.14	18,603	0.23	3,612	0.05
2.75	4,385	0.06	20,711	0.26	1,321	0.02
3	3,612	0.05	15,056	0.19	575	0.01
>3	2,046	0.03	6,431	0.08	150	0.002
Model 1b; total observations = 78,698						
<2	15,122	0.19	1,380	0.02	55,022	0.69
2	18,910	0.24	4,224	0.05	11,632	0.15
2.25	20,990	0.26	9,518	0.12	4,481	0.06
2.5	11,632	0.15	18,910	0.24	3,718	0.05
2.75	4,481	0.06	20,990	0.26	1,369	0.02
3	3,718	0.05	15,350	0.19	587	0.01
>3	2,119	0.03	6,600	0.08	163	0.002

¹Observations relate to weekly scoring of cows (i.e., cow-week risk periods).

Table 4. Population attributable fraction (PAF) calculated using closed equations and a simulation-based approach using data recorded from 2 UK dairy herds; 724 cows held at the Scotland's Rural College Research and Innovation Centre over an 8-yr period (model 1a and 1b) and 1,040 cow herd in Somerset, UK, over a 44-mo period (model 2)¹

BCS category	Closed calculation				Simulation-based approach					
	N ²	Proportion of total N	Odds	Relative risk	PAF (%)	Number of lameness observations			PAF (%)	
						Median _{baseline} ³	Median _{exp} ⁴	No. of observations attributable to exposure	Median	2.5
Model 1a										
3 ⁵	3,612	0.05	Baseline							
<2	14,762	0.19	0.07	1.27	4.49	3,400	3,929	529	3.10	1.71
2	18,603	0.23	0.06	1.13	2.66	3,675	3,969	297	1.73	0.14
2.25	20,711	0.26	0.06	1.10	2.38	3,869	4,123	256	1.50	-1.31
	Total N for herd: 79,565									
Model 1b										
3 ⁶	3,718	0.09	Baseline							
<2	15,122	0.19	0.009	1.60	8.90	731	1,003	272	7.64	2.81
2	18,910	0.24	0.008	1.38	6.00	704	905	201	5.58	1.05
2.25	20,990	0.26	0.007	1.23	4.68	719	861	142	3.93	-0.76
	Total N for herd: 78,698									
Model 2										
1-60 d; BCS >2	7,525	0.52	Baseline							
1-60 d; BCS <2	2,935	0.20	0.11	1.47	9.83	428	578	150	7.49	4.03
61-120 d; BCS >2	2,102	0.14	Baseline							
61-120 d; BCS <2	2,789	0.19	0.09	1.30	5.92	373	458	85	4.28	0.64
	Total N for herd: 14,530									

¹Only results where BCS categories were significant (95% credible intervals for odds ratios did not include 1.00) have been reported in this table.²N = number of observations (observations relate to the following: for herd A, weekly risk periods for each cow, and for herd B, consecutive 30-d risk periods for each cow).³Median_{baseline} = median number of lameness observations for the baseline scenario.⁴Median_{exp} = median number of lameness observations for the exposed scenario.⁵BCS 3 wk previous to lameness events.⁶BCS 1 wk previous to lameness events.

Table 5. Population attributable fraction (PAF) for BCS and previous lameness estimated by simulation-based approach using data recorded from 2 UK dairy herds; 724 cows held at the Scotland's Rural College Research and Innovation Centre over an 8-yr period (herd A) and 1,040-cow herd in Somerset over a 44-mo period (herd B)¹

Scenario		No. of lameness observations ²			PAF (%)		
		Median _{baseline} ³	Median _{exp} ⁴	N _{exp} ⁵	Median	2.5	97.5
Herd A							
Model 1a	BCS gain	17,110	16,510	−600	−3.54	−5.86	−1.28
	BCS loss	17,110	18,140	1,030	5.99	3.36	8.74
	No previous lameness	17,110	3,304	−13,806	−80.69	−79.01	−82.26
Model 1b	BCS gain	3,571	3,282	−289	−8.06	−13.12	−2.22
	BCS loss	3,571	3,968	397	11.20	5.52	17.33
	No previous lameness	3,571	759	−2,812	−78.75	−76.40	−80.98
Herd B							
Model 2	No previous lameness	1,998	346	−1,652	−82.69	−79.28	−85.61

¹Where the exposure has a protective effect, the PAF is reported as negative [e.g., a gain in BCS reduces the risk of lameness (see Table 1)] and therefore this exposure will result in fewer lameness events.

²Observations relate to the following: for herd A, weekly risk periods for each cow, and for herd B, consecutive 60-d risk periods for each cow.

³Median_{baseline} = median number of lameness observations for the baseline scenario.

⁴Median_{exp} = median number of lameness observations for the exposed scenario.

⁵N_{exp} = number of observations attributable to exposure.

Estimating PAF for BCS and Previous Lameness

Results of the scenarios investigated are presented in Table 5.

BCS. A gain in BCS of 0.5, in cows BCS 3 or less, across the whole herd for the 8 yr of data available for herd A resulted in a reduction of 600 predicted lameness events, where the outcome was LS 3, 4, or 5 (model 1a). The median PAF (95% credible interval) for this change in exposure was −3.54% (−5.86 to −1.28%); that is, 3.54% of lameness events in the herd may be avoidable if all cows with BCS <3 in the 3 wk previously were exposed to a 0.5 gain in BCS. When the lameness severity threshold was 2 consecutive wk LS 4 or 5 (model 1b), a greater reduction occurred in lameness events with a median PAF of −8.06% (−13.12 to −2.22%). A loss in 0.5 BCS across all BCS score categories for model 1a resulted in an additional 1,030 predicted lameness events and the median PAF (95% credible interval) for this exposure was 5.99% (3.36–8.74%); that is, 5.99% of lameness in the herd may be avoidable by not exposing the herd to a loss in BCS of 0.5. The median PAF (95%) for this exposure using model 1b, where lameness severity threshold was increased, was 11.2% (5.52–17.33%).

Previous Lameness. When the effect of exposure to all previous lameness events was removed across the whole herd, the predicted number of lameness observations was reduced by 13,806 observations for herd A where the outcome was LS 3, 4, or 5 (model 1a) and 2,812 observations where outcome was LS 4 or 5 on 2 consecutive weeks (model 1b). Of the predicted lameness events, 80.69% (79.01–82.26%) and 78.75% (76.40–80.98%) were attributable to exposure to previ-

ous lameness events over the study period for these 2 outcomes in herd A (model 1a and 1b), respectively. When the effect of exposure to previous lameness was removed across the whole herd in herd B (model 2), the predicted number of lameness events was reduced by 1,652 events; 82.69% (79.28–85.61%) of lameness treatments were attributable to previous lameness over the study period in herd B.

When PAF was estimated for lameness events that occurred at least 5 wk previously, the median (95% credible interval) PAF were 58.97% (56.11–61.67%), 41.67% (36.90–46.19%), and 46.31% (42.08–50.14%), respectively, for models 1a, 1b, and 2.

When PAF was estimated for lameness events that occurred at least 16 wk previously, the median (95% credible interval) PAF were 9.34% (5.14–13.58%), 11.36% (5.49–17.09%), and 21.07% (16.30–25.50%), respectively, for models 1a, 1b, and 2.

DISCUSSION

Previous Lameness Events

This is the first study to quantify the PAF of previous lameness events in cattle on herd level lameness. Estimates of PAF for the 2 herds suggested that between 79 and 83% of lameness was attributable to exposure to previous lameness events (regardless of when they occurred), indicating that this is an important risk factor. When the effect of lameness events that occur >4 and >16 wk previously were investigated, the contribution from previous lameness decreased markedly, although it was still considerable. This finding suggests that lameness might last for some duration (as shown in Figure

2) or that cows can take a considerable amount of time to recover, but that some do fully recover. It appears from these results that a large proportion of the total lameness events in these herds are accounted for by an accumulation of repeat cases. Across the 2 herds, between 52 and 93% of cows were ever lame during their respective study periods, indicating that significant resources are going into treating a large number of lameness cases.

The challenge therefore is to understand why repeat cases are occurring and how to prevent them. The number of repeat lameness events could be influenced by the duration of time individual animals spend within the herd and therefore if cows are not culled for being lame they may experience a higher number of repeat lameness events. It is also possible that some other environmental or animal-based factors could explain a high number of repeat lameness events in certain cows. For example, there may be an interaction between previous lameness and the environment that influences whether cows will go on to have repeated lameness events. It may also be important to prevent the occurrence of the first lifetime lameness event, although based on this analysis it is not possible to know whether it was the first lifetime lameness event or some other environmental or animal-based interaction that is important in consigning a cow to repeat lameness events. In addition, findings from this study highlight that early and effective treatment of lameness reducing the likelihood of recurrence or cases becoming chronic (Thomas et al., 2015) may also be crucial to lameness control at a herd level.

It is widely reported that lameness events increase the risk of future lameness events occurring (Hirst et al., 2002; Green et al., 2014; Randall et al., 2015). Hirst et al. (2002) investigated the relationship between lameness in heifers and the association with future risk, reporting a positive association between claw horn lesions and future risk. These findings were similar to those reported by Randall et al. (2016); more severe claw horn disruption lesions occurring around the time of first calving were associated with a long-term increased risk of lameness. One hypothesis for this association is that underlying pathology carries over from one lactation to the next, making future cases more likely. The increase in lameness prevalence or risk with increasing parity that is widely reported would support this hypothesis (Barker et al., 2009; Randall et al., 2015; Solano et al., 2015). In addition, Newsome et al. (2016) demonstrated that bone development on the caudal aspect of the distal phalanx at slaughter was positively associated with claw horn lesions during life, providing evidence for underlying pathology being associated with previous lameness. An additional

element to the hypothesis explaining the association between previous and future lameness and increased lameness risk with increasing parity is that hypersensitivity and reduction in pressure pain thresholds may develop as a result of long-term pain associated with lameness. Although poorly understood, it is widely reported in the medical literature that disease can lead to long-term changes in the nociceptive nervous system leading to allodynia (pain associated with nonnoxious stimuli) and hyperalgesia (noxious stimuli causing pain of longer duration and higher intensity than normal; Nielsen and Henriksson, 2007; Latremoliere and Woolf, 2009; Woolf, 2011). Laven et al. (2008) demonstrated a long duration of allodynia associated with lameness even after treatment, highlighting the importance of lameness prevention. When the high prevalence of lesions in heifers reported by Capion et al. (2009) and Maxwell et al. (2015) is considered, this becomes even more significant.

The findings from this study highlight the importance of previous lameness events as a risk factor for lameness and therefore the urgent need for further research to identify how to prevent the occurrence of repeat lameness events.

BCS

The results of this study demonstrated the effect of changing BCS across the whole herd; 4% of all lameness events (1 wk with LS 3, 4, or 5) could potentially be avoidable with exposure to a 0.5 increase in BCS in all cows with BCS <3, whereas 8% of all lameness events may be preventable by avoiding exposure to a loss of 0.5 BCS. These figures increased to 6 and 11%, respectively, when the lameness severity threshold was increased. Previous studies have demonstrated that BCS is a risk factor for lameness in all ages of dairy cattle (Hoedemaker et al., 2009; Green et al., 2014; Lim et al., 2015). Randall et al. (2015) found that cows with BCS <2 in the previous 3 wk were at greatest risk of lameness in a longitudinal study using the same data set from herd A as in the current study. Similarly, Green et al. (2014) has shown that cows with BCS ≤ 2 were more likely to be treated for lameness in the following 2 and 2 to 4 mo compared with cows BCS >2, using the same data set from herd B as in the current study. However, this is the first study to evaluate the importance of BCS changes at a herd level in terms of its effect on the total amount of lameness in a dairy herd using simulation that accounts for variability. This is an important step forward from identifying BCS as a risk factor for lameness toward quantifying the effect that this risk factor has on the proportion of lameness events in herds that could be prevented if BCS was

altered. Alawneh et al. (2014) calculated population attributable risk for live weight loss using closed equations and demonstrated that the population level effect of a decrease in live weight over the first 50 d in milk was relatively small; a 3% (95% confidence interval = 1–6%) reduction in the incidence risk of lameness was reported if excessive live weight loss was prevented. The effects of BCS reported for each of the scenarios investigated in this study are similarly relatively small compared with the effect of previous lameness events. However, in herds with fewer repeated lameness events, BCS could be relatively more important.

Comparison of Closed and Simulation-Based Approaches for Estimating PAF

Formulas for calculating population attributable risk or fractions have been derived for different epidemiologic designs, including situations where there is more than one exposure level or where confounding factors exist (Benichou, 2001). However, there are limitations in the use of these formulas when applied to more complex scenarios that are often present in real-life situations, which means they are not directly useable in application. Simulation can be useful in addressing these issues by modeling dynamic interactions between individual animals or groups of animals while taking into account factors that may vary within and across levels of influence. Galea et al. (2010) used obesity as an example to demonstrate how traditional analytical approaches, which focus on the isolation of single disease states and causes, have been challenged by the recognition of dynamic and complex interactions of factors influencing disease outcomes. Complex systems dynamic models can offer an alternative approach. Simulation models parameterized using observations from epidemiological data can be used to investigate inputs and outputs of a complex system and therefore become useful as a tool to test different scenarios. The use of simulation for estimating PAF where data have repeated measures is a novel approach to investigating the importance of risk factors for lameness. Therefore, estimates using a closed equation method were compared with those using simulation. In this study the formula applicable for multiple exposure levels was used to calculate the PAF for BCS categories using data from 2 herds. Results using this closed method were compared with the results generated from posterior predictions. Simulation methods estimated PAF values that were within the 95% credible interval for PAF estimated using closed methods. These results illustrate that simulation-based approaches produce similar, although slightly more conservative, estimates of PAF. As simulation methods account for the variability and can propagate this

through the model to be included in the posterior predictions, the simulation-based results may be the more realistic figure for PAF.

Study Limitations and Generalizability

The main findings of this study were demonstrated in 2 UK herds with different methods of lameness detection. The PAF of comparable scenarios were similar in both herds, giving an indication for possible generalizability of these findings to herds with similar management systems. Although it should be recognized that the PAF estimates reported here are only applicable to changes in the original exposure distribution in these herds (i.e., in herds with a higher median BCS compared with these study herds), the PAF for changes in BCS may differ to that reported in this study. The mean prevalence of lameness over the study period in herd A for LS 3, 4, or 5 was 21.3%, which is lower than prevalence rates reported in other UK studies (Archer et al., 2010; Barker et al., 2010).

This study only investigated the population-level effects of the risk factors BCS and previous lameness. The effect of other risk factors, including environmental risk factors, should also be quantified to understand how these contribute toward lameness at a herd level compared with the risk factors explored in this study.

CONCLUSIONS

This study quantified the effects of the risk factors BCS and the occurrence of previous lameness events on herd level lameness. A loss in BCS of 0.5 across the herd was estimated to contribute toward 6% of the total number of lameness events (1 wk with LS 3, 4, or 5), indicating that this proportion of total lameness could potentially be avoidable in the herds investigated. When the lameness severity threshold was increased (2 consecutive weeks LS 4 or 5), this figure increased to 11%. By comparison, between 79 and 83% of lameness events were estimated to be attributable to exposure to all previous lameness events and between 9 and 21% attributable to exposure to lameness events that occurred at least 16 wk previously. These findings suggest that repeated lameness events (i.e., an accumulation of previous lameness events) contribute toward an overwhelming proportion of the total amount of lameness in the herds investigated. Interactions with environmental or animal-based factors may be important for influencing whether animals go on to have repeated lameness events. Preventing the first case of lameness could potentially be important in avoiding an escalation of repeated lameness events. A novel approach to estimating PAF using simulation enabled complex scenarios to

be investigated while accounting for variability within the herds in this study using longitudinal data with repeated measures.

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REFERENCES

- Alawneh, J. I., M. A. Stevenson, N. B. Williamson, N. Lopez-Villalobos, and T. Otley. 2014. The effects of liveweight loss and milk production on the risk of lameness in a seasonally calving, pasture fed dairy herd in New Zealand. *Prev. Vet. Med.* 113:72–79.
- Archer, S., N. Bell, and J. Huxley. 2010. Lameness in UK dairy cows: A review of the current status. *In Pract.* 32:492–504.
- Barker, Z. E., J. R. Amory, J. L. Wright, S. A. Mason, R. W. Blowey, and L. E. Green. 2009. Risk factors for increased rates of sole ulcers, white line disease, and digital dermatitis in dairy cattle from twenty-seven farms in England and Wales. *J. Dairy Sci.* 92:1971–1978.
- Barker, Z. E., K. A. Leach, H. R. Whay, N. J. Bell, and D. C. J. Main. 2010. Assessment of lameness prevalence and associated risk factors in dairy herds in England and Wales. *J. Dairy Sci.* 93:932–941.
- Benichou, J. 2001. A review of adjusted estimators of attributable risk. *Stat. Methods Med. Res.* 10:195–216.
- Bergsten, C., E. Telezhenko, and M. Ventorp. 2015. Influence of soft or hard floors before and after first calving on dairy heifer locomotion, claw and leg health. *Animals (Basel)* 5:662–686.
- Bicalho, R. C., V. S. Machado, and L. S. Caixeta. 2009. Lameness in dairy cattle: A debilitating disease or a disease of debilitated cattle? A cross-sectional study of lameness prevalence and thickness of the digital cushion. *J. Dairy Sci.* 92:3175–3184.
- Capion, N., S. M. Thamsborg, and C. Enevoldsen. 2009. Prevalence and severity of foot lesions in Danish Holstein heifers through first lactation. *Vet. J.* 182:50–58.
- Chagunda, M. G. G., D. A. M. Römer, and D. J. Roberts. 2009. Effect of genotype and feeding regime on enteric methane, non-milk nitrogen and performance of dairy cows during the winter feeding period. *Livest. Sci.* 122:323–332.
- Galea, S., M. Riddle, and G. A. Kaplan. 2010. Causal thinking and complex system approaches in epidemiology. *Int. J. Epidemiol.* 39:97–106.
- Galindo, F., and D. M. Broom. 2000. The relationships between social behaviour of dairy cows and the occurrence of lameness in three herds. *Res. Vet. Sci.* 69:75–79.
- Gelman, A. 2000. Diagnostic checks for discrete data regression models using posterior predictive simulations. *Appl. Stat.* 49:247–268.
- Gelman, A., X. Meng, and H. Stern. 1996. Posterior predictive assessment of model fitness via realized discrepancies. *Stat. Sin.* 6:733–807.
- Goldstein, H. 2003. *Multilevel Statistics Models*. 3rd ed. Arnold, London, UK.
- Green, L. E., J. N. Huxley, C. Banks, and M. J. Green. 2014. Temporal associations between low body condition, lameness and milk yield in a UK dairy herd. *Prev. Vet. Med.* 113:63–71.
- Green, M. J., P. R. Burton, L. E. Green, Y. H. Schukken, A. J. Bradley, E. J. Peeler, and G. F. Medley. 2004. The use of Markov chain Monte Carlo for analysis of correlated binary data: Patterns of somatic cells in milk and the risk of clinical mastitis in dairy cows. *Prev. Vet. Med.* 64:157–174.
- Hanley, J. A. 2001. A heuristic approach to the formulas for population attributable fraction. *J. Epidemiol. Community Health* 55:508–514.
- Hirst, W. M., R. D. Murray, W. R. Ward, and N. P. French. 2002. A mixed-effects time-to-event analysis of the relationship between first-lactation lameness and subsequent lameness in dairy cows in the UK. *Prev. Vet. Med.* 54:191–201.
- Hoedemaker, M., D. Prange, and Y. Gundelach. 2009. Body condition change ante- and postpartum, health and reproductive performance in German Holstein cows. *Reprod. Domest. Anim.* 44:167–173.
- Hosmer, D. W., and S. Lemeshow. 1989. *Applied Logistic Regression*. Wiley, New York, NY.
- Hudson, C. D., J. N. Huxley, and M. J. Green. 2014. Using simulation to interpret a discrete time survival model in a complex biological system: fertility and lameness in dairy cows. *PLoS One* 9:e103426.
- Latremoliere, A., and C. J. Woolf. 2009. Central sensitization: A generator of pain hypersensitivity by central neural plasticity. *J. Pain* 10:895–926.
- Laven, R. A., K. E. Lawrence, J. F. Weston, K. R. Dowson, and K. J. Stafford. 2008. Assessment of the duration of the pain response associated with lameness in dairy cows and the influence of treatment. *N. Z. Vet. J.* 56:210–217.
- Lim, P. Y., J. N. Huxley, J. A. Willshire, M. J. Green, A. R. Othman, and J. Kaler. 2015. Unravelling the association between lameness and body condition score in dairy cattle using a multistate modelling approach. *Prev. Vet. Med.* 118:370–377.
- Lunn, D., D. Spiegelhalter, A. Thomas, and N. Best. 2009. The BUGS project: Evolution, critique and future directions (with discussion). *Stat. Med.* 28:3049–3067.
- Manson, F. J., and J. D. Leaver. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. *Anim. Prod.* 47:185–190.
- Maxwell, O. J., C. D. Hudson, and J. N. Huxley. 2015. Effect of early lactation foot trimming in lame and non-lame dairy heifers: A randomised controlled trial. *Vet. Rec.* 177:100.
- Mulvany, P. M. 1977. A body condition scoring technique for use with British Friesian cows. *Anim. Prod.* 24:157–158.
- Newsome, R., M. J. Green, N. J. Bell, M. G. G. Chagunda, C. M. Mason, C. J. Sturrock, H. R. Whay, and J. N. Huxley. 2016. Linking bone development on the caudal aspect of the distal phalanx with lameness during life. *J. Dairy Sci.* 99:4512–4525.
- Nielsen, L. A., and K. G. Henriksson. 2007. Pathophysiological mechanisms in chronic musculoskeletal pain (fibromyalgia): The role of central and peripheral sensitization and pain disinhibition. *Best Pract. Res. Clin. Rheumatol.* 21:465–480.
- Pryce, J. E., B. L. Nielsen, R. F. Veerkamp, and G. Simm. 1999. Genotype and feeding system effects and interactions for health and fertility traits in dairy cattle. *Livest. Prod. Sci.* 57:193–201.
- R Core Team. 2016. *R: A language and environment for statistical computing*. R Foundation for Statistical Computing, Vienna, Austria.
- Rabash, J., C. Charlton, W. J. Browne, M. Healy, and B. Cameron. 2009. *MLwiN Version 2.1*. Centre for Multilevel Modelling, University of Bristol, Bristol, UK.
- Randall, L. V., M. J. Green, M. G. Chagunda, C. Mason, L. E. Green, and J. N. Huxley. 2016. Lameness in dairy heifers; impacts of hoof lesions present around first calving on future lameness, milk yield and culling risk. *Prev. Vet. Med.* 133:52–63.
- Randall, L. V., M. J. Green, M. G. Chagunda, C. Mason, S. C. Archer, L. E. Green, and J. N. Huxley. 2015. Low body condition predisposes cattle to lameness: An 8-year study of one dairy herd. *J. Dairy Sci.* 98:3766–3777.
- Rockhill, B., B. Newmand, and C. Weinberg. 1998. Use and misuse of population attributable fractions. *Am. J. Public Health* 88:15–19.
- Solano, L., H. W. Barkema, E. A. Pajor, S. Mason, S. J. LeBlanc, J. C. Zaffino Heyerhoff, C. G. Nash, D. B. Haley, E. Vasseur, D. Pellerin, J. Rushen, A. M. de Passille, and K. Orsel. 2015. Prevalence of lameness and associated risk factors in Canadian Holstein-Friesian cows housed in freestall barns. *J. Dairy Sci.* 98:6978–6991.

Steenland, K., and B. Armstrong. 2006. An overview of methods for calculating the burden of disease due to specific risk factors. *Epidemiology* 17:512–519.

Thomas, H. J., G. G. Miguel-Pacheco, N. J. Bollard, S. C. Archer, N. J. Bell, C. Mason, O. J. Maxwell, J. G. Remnant, P. Sleeman, H. R. Whay, and J. N. Huxley. 2015. Evaluation of treatments for claw horn lesions in dairy cows in a randomized controlled trial. *J. Dairy Sci.* 98:4477–4486.

Woolf, C. J. 2011. Central sensitization: Implications for the diagnosis and treatment of pain. *Pain* 152(Suppl.):S2–S15.

APPENDIX

Appendix Table A1 is supplementary to Table 1.

Table A1. Final logistic regression model (model 1a) with the outcome lameness events based on locomotion scores (LS) ≥ 3 for all covariates other than BCS and previous lameness events, which are reported in Table 1

Intercept	Coefficient: -3.433		
Variable	Odds ratio	Lower 95% CrI ¹	Upper 95% CrI
Weeks in milk (WIM)			
WIM ¹	1.031	1.00	1.01
WIM ²	1.00	1.00	1.00
Parity			
1	Baseline		
2	1.10	1.03	1.17
3	1.46	1.37	1.57
4 +	2.09	1.92	2.28
Age at first calving (mo)			
<25	Baseline		
24 to 27	1.27	1.18	1.35
28 to 30	1.59	1.45	1.74
31 to 33	1.28	1.08	1.53
>33	2.32	1.93	2.79
Feed-genetic group ²			
LF:C	Baseline		
LF:S	1.03	0.97	1.10
HF:C	0.69	0.65	0.74
HF:S	0.67	0.63	0.72
Dry:C	0.68	0.61	0.76
Dry:S	0.69	0.61	0.78
Other:C	2.14	1.75	2.63
Other:S	2.97	2.40	3.67
LS assessor			
1	Baseline		
2	0.43	0.40	0.46
3	0.80	0.74	0.86
4	0.89	0.82	0.96
5	0.77	0.60	0.99
Milk yield 3 wk previously (kg)			
<12	Baseline		
12 to 24	0.88	0.74	1.05
25 to 37	0.80	0.67	0.96
38 to 50	0.77	0.64	0.93
>50	0.65	0.52	0.82
BW 3 wk previously (kg)			
<550	Baseline		
500 to 700	0.89	0.84	0.96
>500	0.81	0.74	0.90
BCS change 0 to 4 wk postcalving			
No change	Baseline		
Loss ≥ 0.25	1.13	1.04	1.24
Gain ≥ 0.25	1.08	0.95	1.24
Logarithm week ³ (LOGwk)			
LOGwk ¹	1.37	1.31	1.44
LOGwk ²	1.35	1.29	1.40
LOGwk ³	1.04	1.03	1.05

¹CrI = credible interval.

²Feed-genetic groups include low forage (LF), high forage (HF), control (C), and select (S). Dry refers to dry cows and other refers to all other management groups outside of LF, HF, and dry.

³Week refers to week of the study period.